CARDIOPULMONARY EFFECTS OF URINARY BLADDER DISTENTION

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It is a pleasure to acknowledge the assistance of Dr. Byong M. Kim who worked with me on many of these experiments.

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INTRODUCTION:

These experiments began following observation of a 70 year old patient whose chronic lung disease and congestive heart failure were in good control until he developed acute urinary obstruction associated with benign prostatic hypertrophy. Severe dyspnea developed but ceased after catheterization for relief of bladder distention. The catheter was removed and within 24 hours the patient's dyspnea recurred. Despite absence of an urge to urinate, he was again found to have an overfull bladder. Once more, he was catheterized, and again coincident with relief of his urinary obstruction, his cardiorespiratory status improved.

It thus became evident that for some reason bladder distention was a life-threatening situation for this patient. Having observed this intriguing sequence of events, I decided to investigate, in dogs, the possibility that urinary bladder distention caused cardiopulmonary changes capable of producing dyspnea.

HISTORY:

Dyspnea is the subjective sensation of difficulty in breathing. The mechanism by which respiratory discomfort is perceived is unknown, but an emotional factor is probable since the severity of the symptom often correlates poorly with the degree of respiratory impairment present (1). Several theories have been proposed to explain the etiology of dyspnea.

The oldest is the concept that chemical changes in the blood involving increased carbon dioxide concentration, decreased oxygen concentration or decreased pH produce dyspnea by stimulating the respiratory center in the brain, or the carotid body. Although such alterations do increase the rate and depth of respiration, they fail to explain the frequent occurrence of dyspnea in patients whose blood gases are normal, or nearly so (2).

According to another explanation of dyspnea, because of disease, mechanical interference with respiration puts an excessive work load on the respiratory muscles. Muscular fatigue is thought to ensue and to be the direct cause of dyspnea (13).

Wright pictures breathing as being controlled by a balance of inspiratory impulses originating in the respiratory center and opposing inhibitory impulses, arising from stretch receptors triggered by inflation of the lung. The latter are carried to the respiratory center by the vagus nerve. If the lung is unable to inflate normally, there is inadequate stretch receptor discharge resulting in decreased inhibitory response. The consequent dominance of the inspiratory drive causes the sensation of breathlessness (4).

Another theory is based on Campbell's investigations of respiratory proprioceptive mechanisms. He postulates that receptors in the

lung or thoracic cage detect changes in pressure, volume and flow and thereby relay to the consciousness information as to the amount of ventilation achieved. A conscious perception of inability of the actual ventilation to meet the ventilatory demands of the respiratory center may, therefore, be responsible for dyspnea (1,5).

In 1934 Christie and Meakins reported that dyspneic patients in congestive heart failure had decreased distensibility of their lungs and that improvement in distensibility was associated with disappearance of the dyspnea (6). This finding, while not revealing the mechanism by which dyspnea occurs, did define a specific type of respiratory impairment which produced the symptom.

The last three of the four theories of the etiology of dyspnea previously discussed are all compatible with the observation that the lungs of dyspneic patients may be less distensible than normal. The patient with stiffened lungs must exert extra pressure to provide adequate alveolar ventilation. To do this he must work harder with his respiratory muscles and muscular fatigue is likely to appear. It is also possible that decreased lung distensibility may be associated with decreased stretch receptor discharge and thereby result in either a decreased involuntary inhibitory respiratory response or a message to the consciousness indicating decreased ventilation in relation to the demands of the respiratory center.

One can say, therefore, that although knowledge of the mechanism of dyspnea is limited, several provocative theoretical explanations have been proposed and a distinct type of cardiopulmonary abnormality, namely a stiffened lung, has been observed to be often present in the dyspneic patient. As to the possible relation of urinary bladder distention to dyspnea, a number of scientific reports are pertinent.

For many years it has been known that stimulation of abdominal viscera may give rise to circulatory or respiratory phenomena. In 1899 Sherrington, while studying the afferent nerves of the viscera, found that the mechanical distention transiently produced by injecting saline into the bile duct of anesthetized mammals evoked an immediate rise in blood pressure (7). During the next 35 years various authors reported changes in heart rate, blood pressure, electrocardiogram, or respiratory rate due to manipulation of the gall bladder and intestines (8,9,10).

In 1937 Talaat was investigating the sensory innervation of the urinary bladder, when he incidently discovered that vesical distention resulted in increased blood pressure in anesthetized animals (11). Watkins in 1938 confirmed this work and found that the rise in blood pressure correlated quantitatively with bladder pressure but not with bladder volume (12).

More recently it was noted that, during cystometrographic examinations, patients with high traumatic transections of the spinal cord were subject, at high vesical pressures, to sudden increase in blood pressure, slowing of pulse rate, change in electrocardiogram, and localized cutaneous vasodilation. The type and extent of these alterations varied with the site of the cord injury (13). In 1960 Agrest and Roncoroni studied two patients with paraplegia secondary to traumatic damage of the spinal cord and found that bladder distention caused both pulmonary and femoral artery pressures to rise, without affecting cardiac output or central blood volume (14).

A study performed on healthy women in the U.S.S.R. revealed slight changes in the electrocardiogram when the subjects' bladders were distended with fluid volumes less than those at which there was a conscious urge to urinate (15). Berman and Rose studied intact anesthetized dogs

in 1958 and failed to find significant cardiovascular alterations in response to bladder inflation (16). These authors rapidly injected large amounts of saline under very high pressure into bladders and found no changes in the electrocardiogram or the aortic and right ventricular pressure.

THEORETICAL CONSIDERATIONS:

The measurement of the distensibility of the lungs is called compliance. It determines the strain produced by a given stress and expresses a relationship between force and stretch, or its equivalent, a relationship between pressure and volume. If a known volume of air is injected into a balloon and the resultant pressure rise inside the balloon measured, one knows the compliance of the balloon. Similarly, to obtain total compliance of the lung a measured quantity of air is forced in and the intra-alveolar pressure rise noted (1, 17). If the pressure rise is abnormally high for the volume injected, the compliance is decreased.

Since reduction in compliance has been implicated as a cause of dyspnea, the hypothesis was made that it was responsible for the respiratory distress associated with bladder distention. To partially test the hypothesis experimentally, I proposed to study the effect of distending a dog's bladder on his lung compliance.

Cardiopulmonary changes reflect many complexly interrelated factors and to illuminate even dimly the nature of such changes it is necessary to investigate several areas rather than settle for a single parameter such as compliance. To further define physiological alterations induced by urinary bladder distention, studies were made of pulmonary artery and aortic pressures, cardiac output, and arterial blood gases.

METHODS:

All experiments consisted of control studies on anesthetized animals with emptied bladders followed by studies in the same animals while their bladders were distended. Anesthesia eliminates the possibility of a psychic factor and previous work suggests that neither anesthesia nor the trauma of experimental procedures should significantly influence the results during the period of the experiment (18).

Eleven male dogs weighing between 26 and 46 lbs. were anesthetized with 60 mgs. of intravenous pentobarbital per five lbs. body weight. In another dog (#3) intravenous Dial with Urethane, .5 ml. per kg. body weight,was used as anesthetic. Tracheotomies were performed and the tracheotomy tubes connected to a Palmer respirator pump, adjustable for rate and tidal colume.

For total thoracic compliance in seven dogs the airway was switched from the Palmer Pump at end-expiration to a closed three way circuit with a water manometer and a specially constructed device for injection of measured quantities of air (Figure 1). A series of measured volumes of air was quickly forced into the lungs and the pressure exerted against the manometer at each volume recorded. At least five injections up to a maximum total injected volume of 798 cc. were used for each compliance curve.

In five other dogs the presence or absence of compliance changes was inferred from the pulmonary artery pressure recording. When an individual breathes in and out, there are concomitant changes in intrathoracic pressure which usually cause slight alterations of the pulmonary artery pressure during the respiratory cycle. If compliance decreases, the intrathoracic pressure changes will be greatly accentuated and their effects on the pulmonary artery pressure will be more noticeable. Therefore,

if during a control study a dog showed little or no respiratory effect on the pulmonary artery pressure, but with a distended bladder had noticeably increased respiratory excursions of the pressure, a reduction in compliance was assumed.

For cardiac output and aortic pressure determinations, a femoral artery and vein cutdown was performed and polyethylene catheters inserted through these vessels into the inferior vena cava and aorta respectively. To get pulmonary artery pressures, a cutdown was done on the right external jugular vein and under fluoroscopic visualization a radiopaque polyethylene catheter inserted through the vein and the right side of the heart into the artery.

Cardiac outputs were estimated by the method of Kinsman et al. with Cardio-Green, a tricarbocyanine dye, as an indicator and inscription of a dye dilution curve by means of a cuvette densiometer manufactured by Guilford Instrument Laboratories (19, 20). For arterial pressures, variable inductance transducers were connected to a blood pressure control unit (Hathaway Type-MBC-2) monitored by an oscilloscope. Pressures and cardiac outputs were recorded on a Hathaway multi-channel recorder (Type S-14-C) with an optical system and negative photographic paper film.

Arterial blood samples were collected anaerobically in heparinized syringes. A Beckman Physiological Gas Analyzer, Model 160, was used to determine pH, oxygen tension (PaO2), and carbon dioxide tension (PaCO2) with a glass pH electrode and Clark and Severinghaus gas electrodes respectively.

Before beginning the control determinations, a polyethylene catheter was inserted through the urethral orifice into the bladder. The bladder was emptied and the catheter connected, via a three way stopcock, to a water manometer for pressure readings. After completion of the control studies, measured volumes of saline were slowly injected into

the catheter and the pressures recorded.

In dogs, as in humans, bladder pressure rises only slightly until a certain volume of intraluminal fluid accumulates. At this point the detrusor muscle contracts and there is a sudden sharp rise in pressure (21). A pressure of 100 mm. of water was arbitrarily selected as indicative of bladder distention and when this pressure was attained the catheter was clamped and reopened at the end of the experiment, when the pressure was checked to be sure a satisfactory level had been maintained. Between 75 and 250 cc. of injected saline were required for this amount of pressure rise. Cardiopulmonary studies during bladder distention were done at least 15 minutes after a satisfactory bladder pressure was established.

RESULTS:

Compliance.

Direct measurements were done in seven dogs. Four showed decreased compliance during bladder distention as compared with the control value. In the other three there was no significant change while the bladder was inflated.

In the four dogs whose compliance decreased significantly (#3, #4, #5, and #9) the calculations in Table I and accompanying graphs (Figures II - VIII) express the resuult of a single control and experimental run representative of several determinations. In the remaining three animals no single curve was clearly representative of control or experimental results and in each case an average of the two technically most satisfactory curves was used.

Rough estimates of compliance were obtained in five other dogs using the pulmonary artery pressure recordings. This was done by dividing the tidal volume by the change in pulmonary artery diastolic pressure during the respiratory cycle. Three of the five dogs showed a decrease in compliance during bladder distention (Table II).

Thus, of the twelve dogs studied, seven had decreased compliance while the bladder was inflated and five demonstrated no significant change. On the basis of these results the twelve dogs were divided into two groups; Group I in which compliance decreased at high vesical pressures and Group II in which no difference was noted. Studies of other parameters of cardiopulmonary function revealed a separate pattern of changes in each group.

Pulmonary Artery Pressure

In Group I all three dogs in which pulmonary artery pressures were obtained had rises in both systolic and diastolic pressures during bladder distention as opposed to the control level (Table III).

In one dog (#11), as shown in Figure IX, the pulmonary artery pressure

increased while the bladder was distended, decreased slightly while the bladder was being emptied, and returned to the control level within five minutes after bladder pressure returned to zero. After deflation the pulmonary artery pressure respiratory excursion which had appeared at the high vesical pressure was no longer present.

In another animal (#15), however, both increased pressure and decreased compliance were still present 10 minutes after relief of bladder distention. No other post-distention studies were done in Group I dogs; instead autopsies were performed in several with the bladder still distended to ensure that the inflation procedure had been properly carried out.

Attempts were made to determine pulmonary artery pressure in two Group II dogs. In one straying of the baseline invalidated the results. In the other the catheter in the pulmonary artery probably slipped into the right ventricle between the control and experimental runs. Although these technical difficulties prohibited quantitative accuracy in determining pulmonary artery pressures the recordings in each case suggested no significant relative change between control and experimental pressures.

Aortic Pressure

In Group I all three dogs in which aortic pressure was measured showed increases in both systolic and diastolic levels during bladder distention. In Group II four aortic pressures were recorded. Two were unchanged and two decreased during bladder distention (Table IV).

Pressures in Dogs #3, #6, and #7 are estimated from an arbitrary baseline value because no calibration was performed. These pressures are accurate, however, for indication of relative change between control and experimental situations.

Nothing was observed at the time of the experiment to explain the low control pressure in Dog #15. Two of the three Group I dogs (#15 and #16) displayed increased respiratory effect on the aortic pressure during bladder distention, similar to that seen in the pulmonary artery pressure.

Heart Rate

Three of four dogs in Group I had an increased heart rate during bladder distention. Rate increased in two of four dogs in Group II (Table V).

In dog #3 Dial with Urethane was used for anesthesia, while all other dogs were anesthetized with pentobarbital. This probably explains the slower heart rate in that animal.

Cardiac Output

Both dogs studied in Group I had increased cardiac outputs during

bladder distention, while circulation time decreased and central blood volume was not appreciably altered. Cardiac output decreased in both Group II dogs in which it was measured (Table VI).

The cardiac outputs in Dogs #16 and #6 are estimated from an arbitrary baseline but accurate as regards comparison of control and experimental values in each dog.

Arterial Blood Studies

In Group I PaO2 increased during the experimental determination, while pH and PaCO2 remained at the control level. In Group II PaO2 increased, pH decreased and PaCO2 did not change (Table VII).

SUMMARY OF CARDIOPULMONARY CHANGES DURING DISTENTION

	Group I	Group II
Compliance	decreased	no change
P. A. Pressure	increased	no change
Aortic Pressure	increased	half decreased half no change
Cardiac Output	increased	decreased
Circulation Time	decreased	one decreased one no change
Central Blood Volume	no change	decreased
Heart Rate	increased	half increased half no change
pH	no change	decreased
PaO2	increased	increased
PaCO2	no change	no change

DISCUSSION:

These studies clearly establish that urinary bladder distention has cardiopulmonary effects in anesthetized dogs. Although all dogs tested did not demonstrate identical changes, there is a striking and convincing similarity in those seen in the Group I dogs. It may be assumed that the cardiopulmonary changes in Group I represent a characteristic physiological response to bladder distention and that for some unknown reason, such as individual variability, inadvertent differences in technique, inadequate sensitivity of measuring devices, or an unrecognized physiological unreadiness, the minority in Group II failed to show the same pattern of response.

Quite likely the observed effects of bladder distention are part of a reflex. Receptors in the bladder may respond to pressure changes and by a neural or humoral mechanism cause cardiopulmonary changes. The negative results of Berman and Rose could have been due to their bladder inflation technique by which saline was injected very rapidly and at such high pressure that in some dogs the bladder burst. Possibly bladder receptors were unable to respond to the infusion either because of the speed of the injection or the extreme and unphysiological pressure under which it was done.

The compliance measured in these experiments is that of both lung and thoracic wall. Since there are concurrent related cardiopulmonary changes, it is most likely that the primary cause of the compliance reduction is decreased compliance of the lung alone. However, without additional data, the possibility cannot be ruled out that the reduced compliance is due to increased tone of the thoracic musculature.

Assuming that lung compliance is reduced, this may be caused by changes in the elastic properties of the lung. There could be changes in the lung tissues or alterations in the fluid coating the alveoli, where altered surface tension may affect lung elasticity (22).

Another factor which can alter compliance is change in lung volume. As a measure of distensibility compliance expresses the effect of a unit of pressure on a unit of surface area. If the surface area is decreased, the compliance is also. When lung volume is reduced a given intrapleural pressure change causes less volume change and the lung is therefore less distensible. Decreased functional residual capacity could, therefore, have been related to the decreased compliance in the Group I dogs, but there is no readily apparent mechanism by which such a change could occur. Increased intra-abdominal pressure from the distended bladder is unlikely in view of the small size of the organ.

Several pathophysiological conditions are commonly associated with low compliance (1). One, fibrosis, cannot be a factor in this acute experiment. Another, pulmonary vascular congestion, is ruled out by absence of change in the central blood volume during bladder distention.

A more likely influence on compliance in these experiments is bronchoconstriction due to spasm, edema or bronchial congestion. While the bladder was inflated, expirations in most Group I dogs appeared to be prolonged, suggesting that increased airway resistance was present.

Cardiac output is determined by heart rate and stroke volume. The increased cardiac output in dog #3 is mostly accounted for by increased heart rate, but there is also a small increase in stroke volume. Increased heart rate is also a factor in Dog #16, but stroke volume, while probably elevated, cannot be properly evaluated since the cardiac output is estimated from an arbitrary baseline. It is possible that the sympathetic nervous system may be involved in these phenomena, since both increased heart rate and increased strength of cardiac contraction are well known effects of sympathetic stimulation (23).

Several physiological factors may cause elevation of the mean pulmonary

artery and aortic pressures. Increased blood viscosity, as in polycythemia for example, causes a rise in blood pressure by increasing resistance to flow. In these studies such a mechanism is unlikely.

Increased cardiac output elevates the blood pressure by pouring an increased volume of blood into the vascular system. Probably the most important cause of a rise in blood pressure is an increased peripheral resistance due to arteriolar narrowing. The blood pressure is equal to the product of the cardiac output times the peripheral resistance (23) and increase in the former or both account for the results obtained in these experiments.

Since cardiac output is increased it must be a factor in the blood pressure rise. Unfortunately, satisfactory simultaneous determinations of true cardiac output and true arterial pressures were not obtained and it is, therefore, impossible to estimate whether pulmonary or systemic vascular resistance changed.

In the two paraplegic patients studied by Agrest and Roncoroni a pulmonary and aortic pressure rise occurred, with no change in cardiac output and a slowing of heart rate, indicating a rise in vascular resistance (14). It is interesting to speculate on the possibility that their patients' neurological lesions may explain the differing response in heart rate and cardiac output and that the other responses are caused by the same mechanism present in the Group I dogs.

Sympathetic discharge can cause a rise in blood pressure as well as increased cardiac output and heart rate. However, it is unlikely to be solely responsible for the changes in Group I, if it is involved at all, since it does not explain the compliance changes.

The significance of the increased PaO2 is difficult to assess. Both groups of dogs showed this change, however the increase in Group I is much greater. Current work in this laboratory suggests that dogs normally have

measurable pulmonary arterio-venous shunting (24). It may be that the increased PaO_2 is due to a decreased amount of shunting of blood during bladder distention.

The possibility that bladder distention may result in cardiopulmonary embarrassment of sufficient severity to cause dyspnea is of practical clinical importance. Not only the studies in dogs just discussed, but also those in paraplegic patients by Agrest and Roncoroni and in healthy humans by Ganelina and Tsvibel are suggestive in this regard (14, 15). Sudden onset of unexplained dyspnea in a patient with a cardiorespiratory disorder should be an indication for study of the urinary tract. Especially when neurological lesions are present or when a patient is depressed or comatose, careful attention should be paid to maintenance of urinary tract patency, not only for reasons related to the tract itself, but also, because bladder distention may exacerbate cardiovascular or pulmonary problems. Indeed, it seems likely that in an extremely ill patient bladder distention could be a lethal complication via the cardiopulmonary route.

SUMMARY:

Cardiopulmonary function was studied in twelve anesthetized dogs before and during urinary bladder distention. Seven of the twelve demonstrated a reduction in total compliance during distention. The group of dogs with decreased compliance also showed increased aortic and pulmonary artery pressures, increased cardiac output without alteration in central blood volume, increased heart rate, and arterial blood gas change. It is concluded that these results establish an effect of bladder distention on cardiopulmonary function and it is suggested that a neural or humoral reflex mechanism is in operation. These changes are of potential clinical significance.

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TABLE I

Total Compliance at Maximum Intrathoracic Pressure

Vol. in ml./Pres. in mm. H_2O

	Control	Experimental	% Change
Dog #3	7.1	5.1	-28.6
Dog #4	8.0	6.2	-22.5
Dog #5	9.4	4.9	-48.0
Dog #6	3.15	3.10	- 1.6
Dog #7	3.91	3.91	0
Dog #9	2.5	2.1	-16.0
Dog #10	2.70	2.65	- 1.8

Change greater than 10% considered to be significant.

TABLE II

Compliance Inferred from Pulmonary Artery Pressure Changes

Vol. in ml./Pres. in mm. Hg.

	Control	Experimental	% Change
Dog #11	.375	.094	-75
Dog #13	.250	.250	0
Dog #14	.033	.037	+12
Dog #15	.079	.012	-85
Dog #16	.035	.016	-54

(If a diastolic pressure change is accurate to $\pm$ 1 mm. Hg., a compliance change greater than 35% is significant).

TABLE III

GROUP I PULMONARY ARTERY PRESSURE

mm. Hg.

	Control	Experimental	Difference
Dog #11	13/3	38/23	+25/+20
Dog #15	22/18	31/24	+ 9/+6
Dog #16	28/14	36/20	+ 8/+6

T A B L E IV

Aortic Pressure

mm. Hg.

Group I	Control	Experimental	Difference
Dog #3	180/95	195/110	+15/+15
Dog #15	75/55	133/105	+58/+50
Dog #16	122/87	150/126	+28/+39
Group II			
Dog #6	120/95	115/95	-5/0
Dog #7	122/95	122/100	0/+5
Dog #13	137/108	113/78	-24/-30
Dog #14	195/140	140/105	-55/-35

T A B L E V

Heart Rate Per Minute

Group I	Control	Experimental	% Change
Dog #3	86	100	+16
Dog #11	210	212	+ 1
Dog #15	171	228	+33
Dog #16	190	199	+ 5
Group II			
Dog #6	210	220	+ 5
Dog #7	164	164	0
Dog #13	185	185	0
Dog #14	133	206	+55

TABLE VI

Cardiac Output and Related Studies

		Cardiac Output l/min.	Circ. Time secs.	C. B. V. ml.
Group I				
Dog #3	Control	1.08	11.3	203
	Experimental	1.32	9.0	198
	% Change	+ 22	- 20	- 2
Dog #16	Control	2.28	5.4	205
	Experimental	2.50	4.8	200
	% Change	+ 10	- 11	- 2
Group II				
Dog #6	Control	2.60	7.9	342
	Experimental	2.00	7.9	263
	% Change	- 23	0	- 23
Dog #7	Control	3.72	6.7	415
	Experimental	2.80	6.4	392
	% Change	- 25	- 4	- 6

T A B L E VII

Arterial Blood Studies

Group I

		Control	Experimental	Difference
Dog #3	pH	7.17	7.18	+ .01
	Pa02	96	114	+ 18
	PaC02	37	40	+ 3
Dog #4	pH	7.23	7.24	+ .01
	Pa02	77	85	+ 8
	PaC02	44	36	- 8
Dog #9	pH	7.56	7.48	+ .08
	Pa02	78	102	+ 24
	PaC02	19	22	+ 3

Group II

		Control	Experimental	Difference
Dog #6	pH	7.40	--	?
	Pa02	84	88	+ 4
	PaC02	30	27	- 3
Dog #7	pH	7.49	7.43	-.06
	Pa02	90	95	+ 5
	PaC02	37	29	- 8
Dog #13	pH	7.44	7.36	- .08
	Pa02	83	85	+ 2
	PaC02	26	27	+ 1

FIGURE I.

SYSTEM FOR MEASUREMENT OF COMPLIANCE

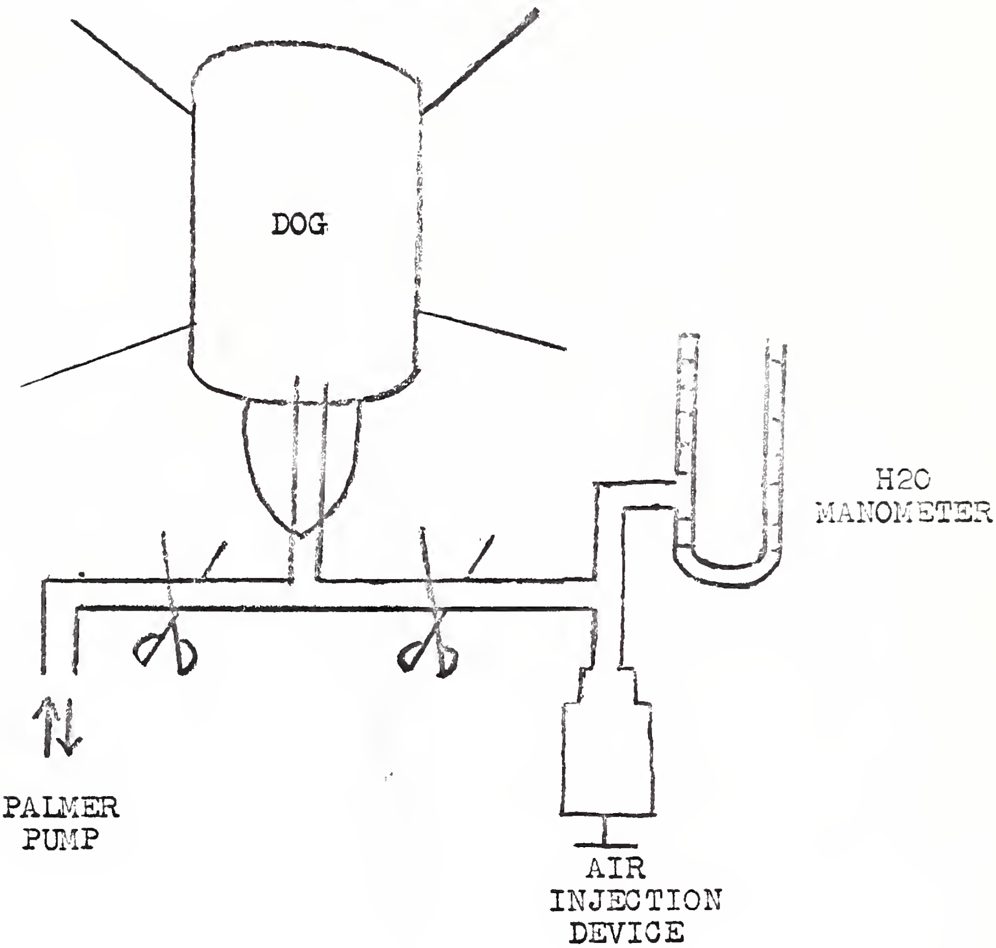
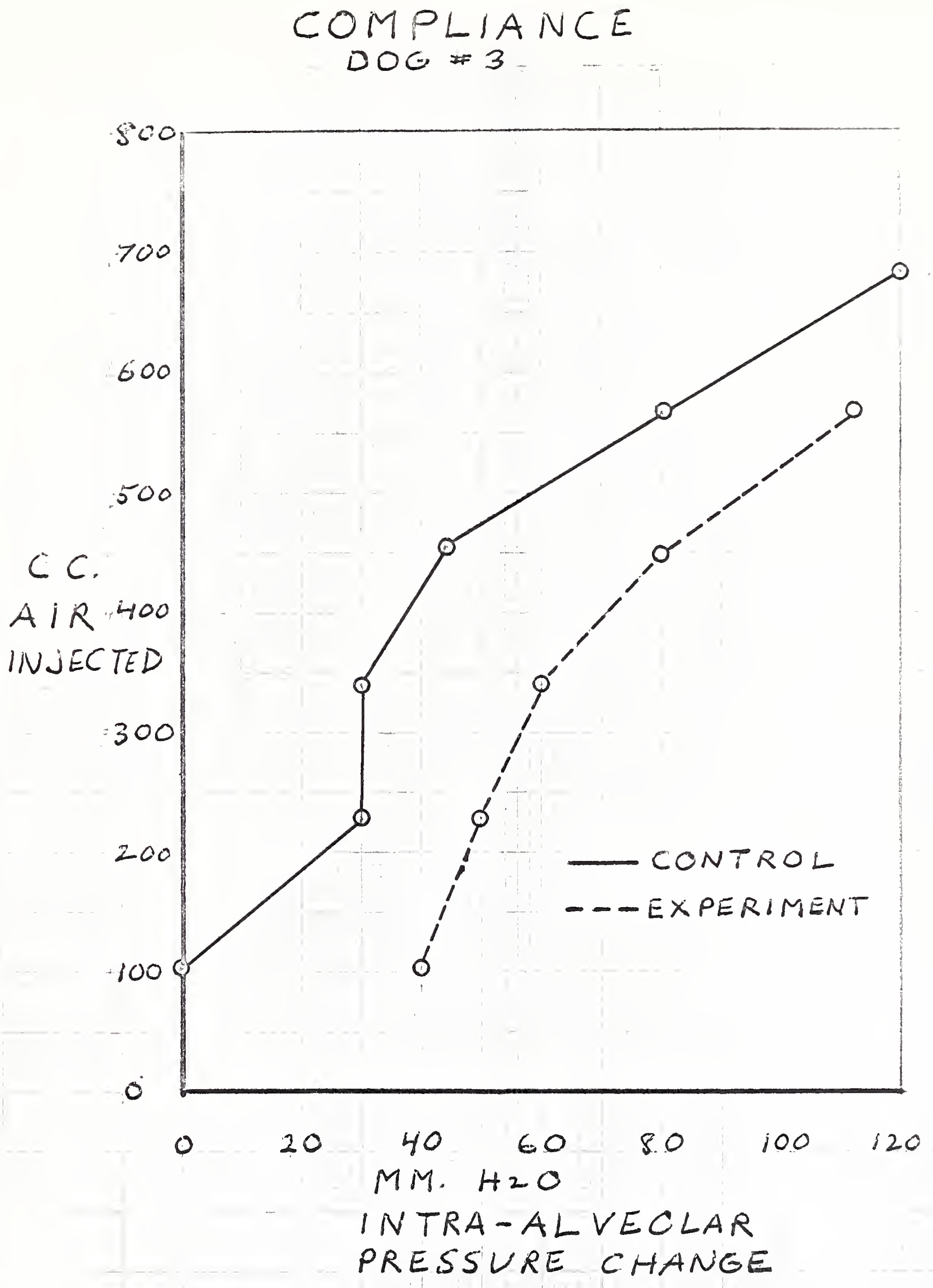


FIGURE II.



COMPLIANCE
DOG #3
800
700
600
500
400
300
200
100
0
C C.
AIR
INJECTED
CONTROL
EXPERIMENT
0
20
40
60
80
100
120
MM. H2O
INTRA-ALVEOLAR
PRESSURE CHANGE

FIGURE III.

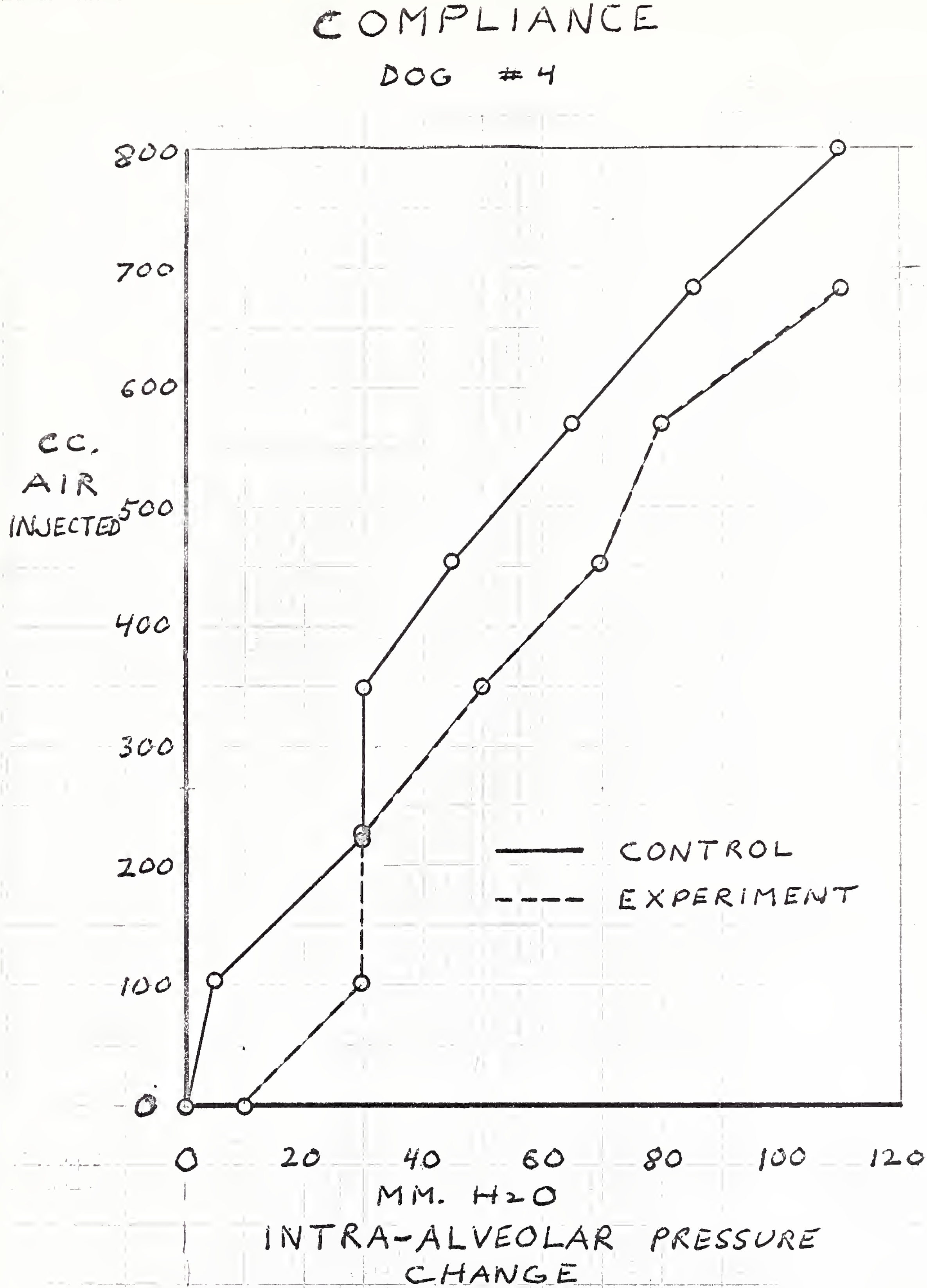
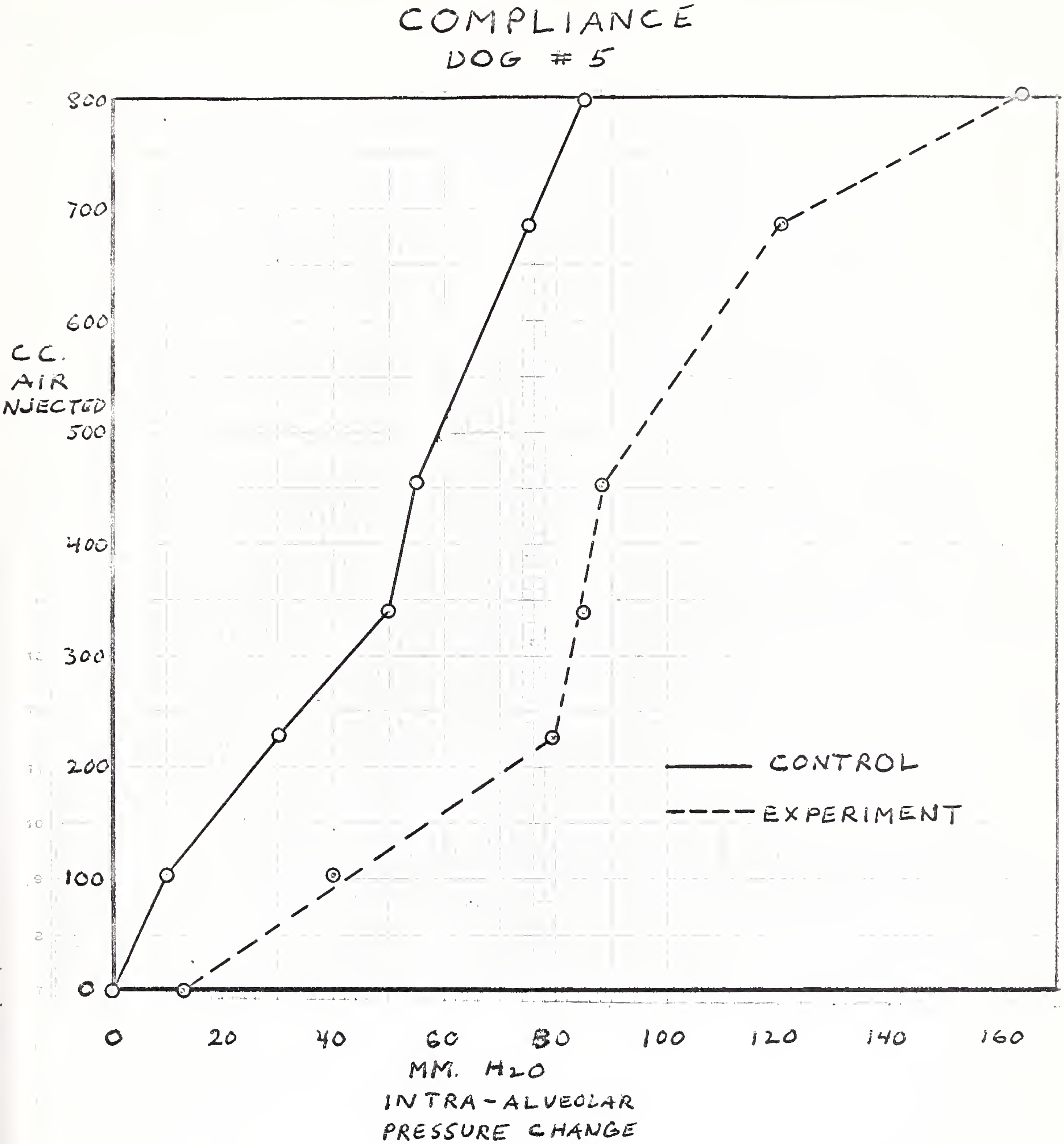
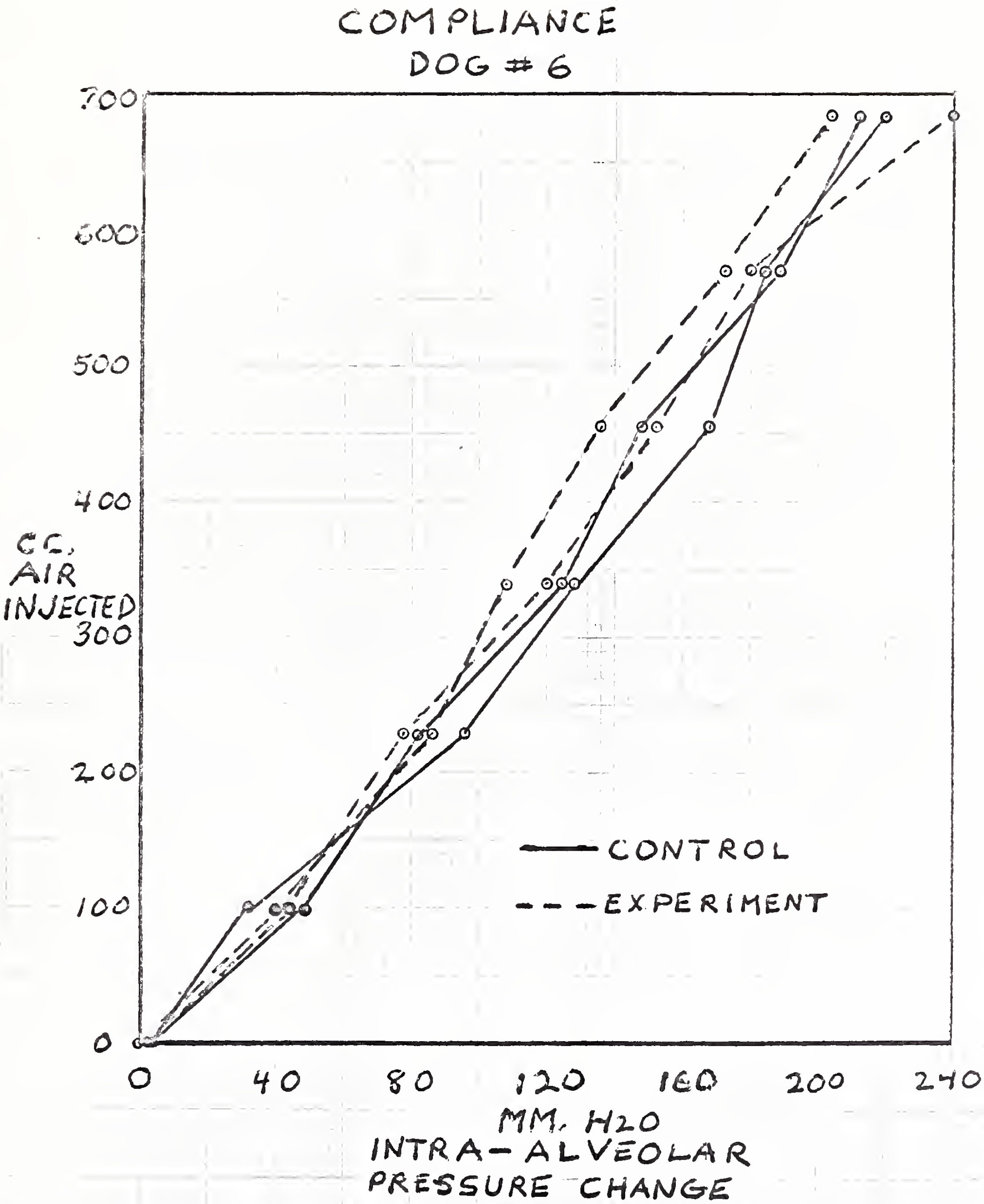

COMPLIANCE
DOG #4
800
700
600
CC.
AIR
INJECTED
500
400
300
200
100
0
CONTROL
EXPERIMENT
0
20
40
60
80
100
120
MM. H2O
INTRA-ALVEOLAR PRESSURE
CHANGE

FIGURE IV.



COMPLIANCE
DOG # 5
C.C.
AIR
NJECTED
800
700
600
500
400
300
200
100
0
0
20
40
60
80
100
120
140
160
MM. H_2O
INTRA-ALVEOLAR
PRESSURE CHANGE
CONTROL
EXPERIMENT

FIGURE V.



COMPLIANCE
DOG # 6
700
600
500
400
CC. AIR INJECTED
300
200
100
0
0
40
80
120
160
200
240
MM. H2O
INTRA-ALVEOLAR PRESSURE CHANGE
CONTROL
EXPERIMENT

FIGURE VI.

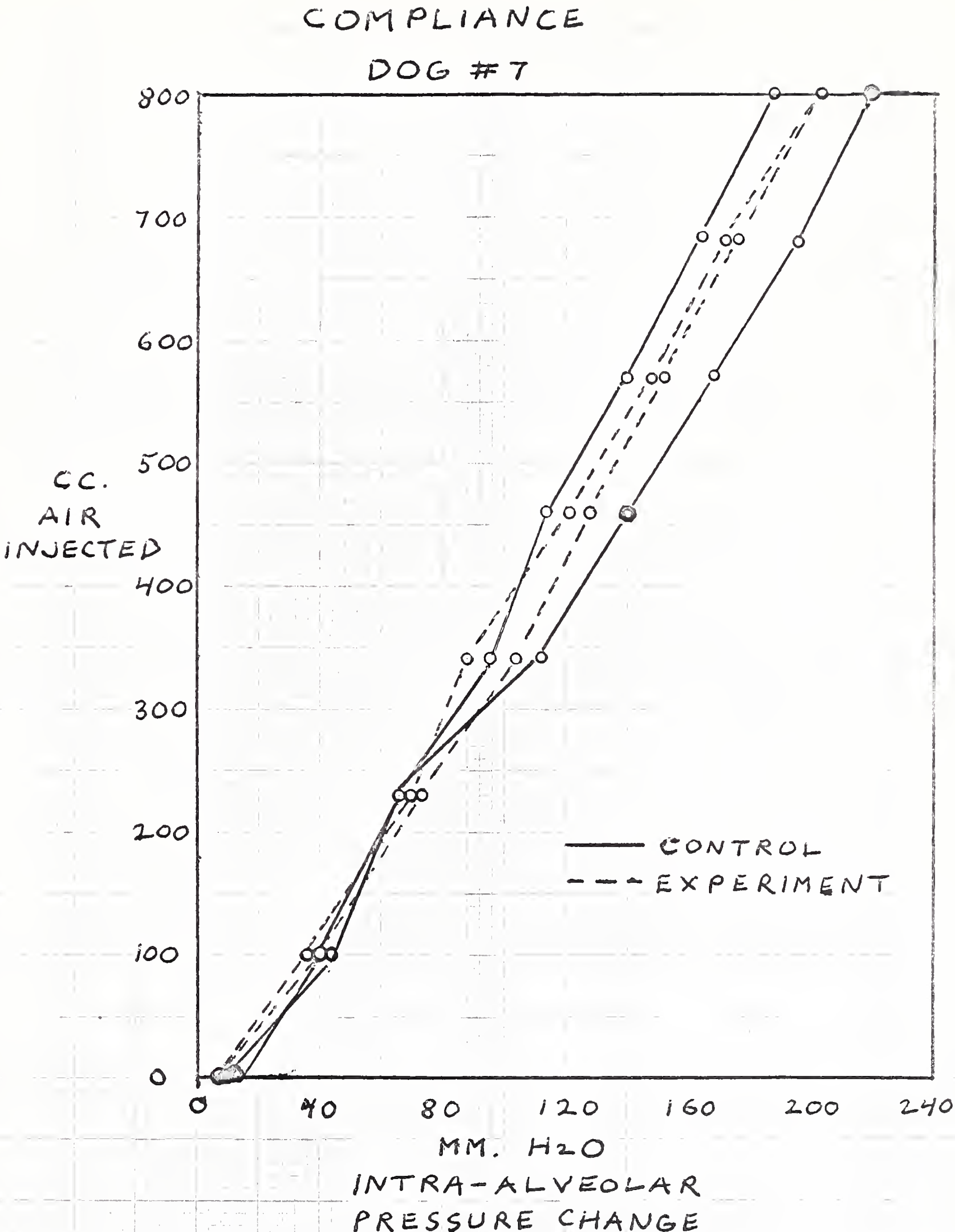
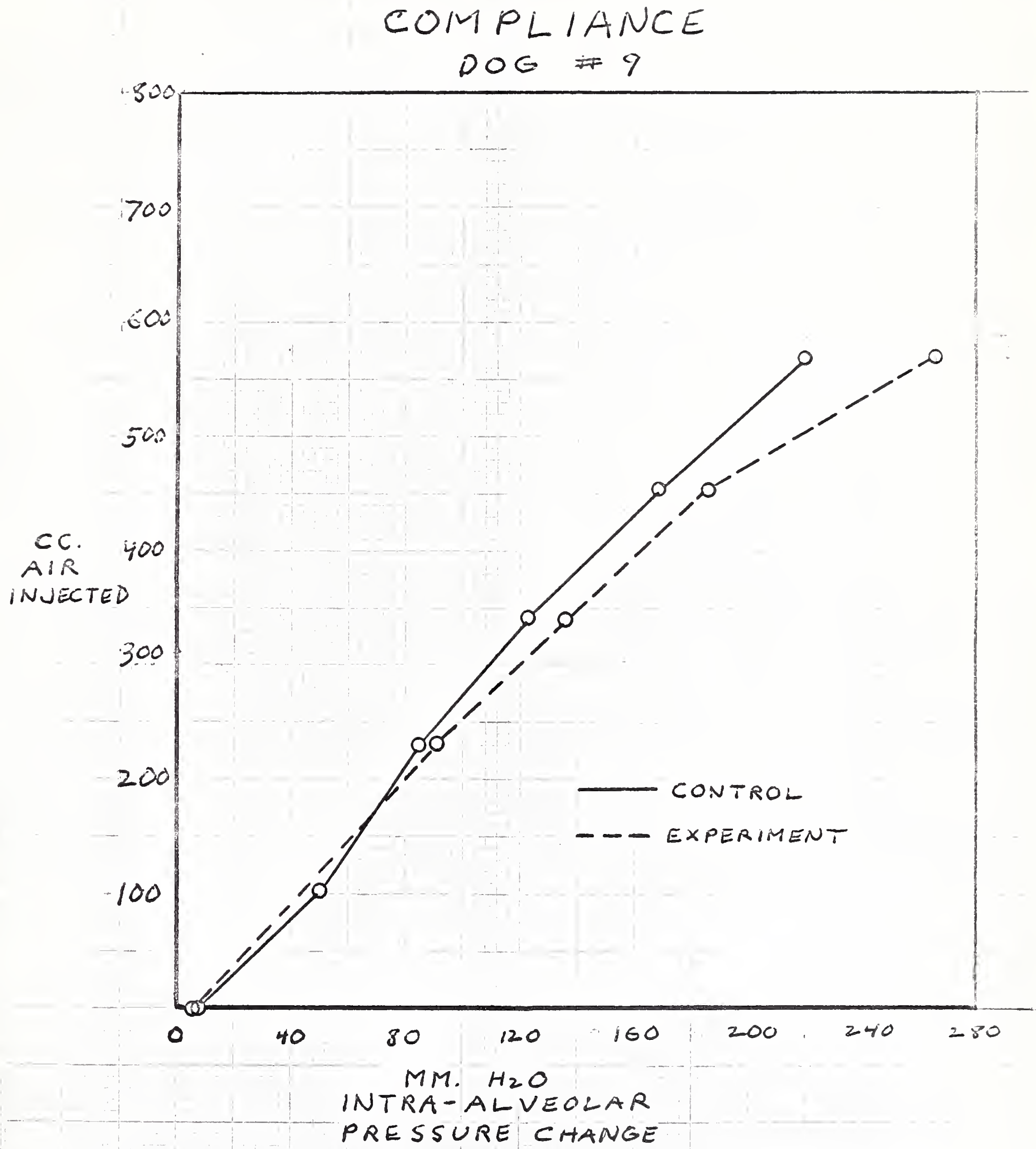

COMPLIANCE
DOG #7
800
700
600
500
400
300
200
100
0
CC.
AIR
INJECTED
0
40
80
120
160
200
240
MM. H_2O
INTRA-ALVEOLAR
PRESSURE CHANGE
CONTROL
EXPERIMENT

FIGURE VII.



COMPLIANCE
DOG # 9
800
700
600
500
400
300
200
100
CC.
AIR
INJECTED
CONTROL
EXPERIMENT
0
40
80
120
160
200
240
280
MM. H_2O
INTRA-ALVEOLAR
PRESSURE CHANGE

FIGURE VIII.

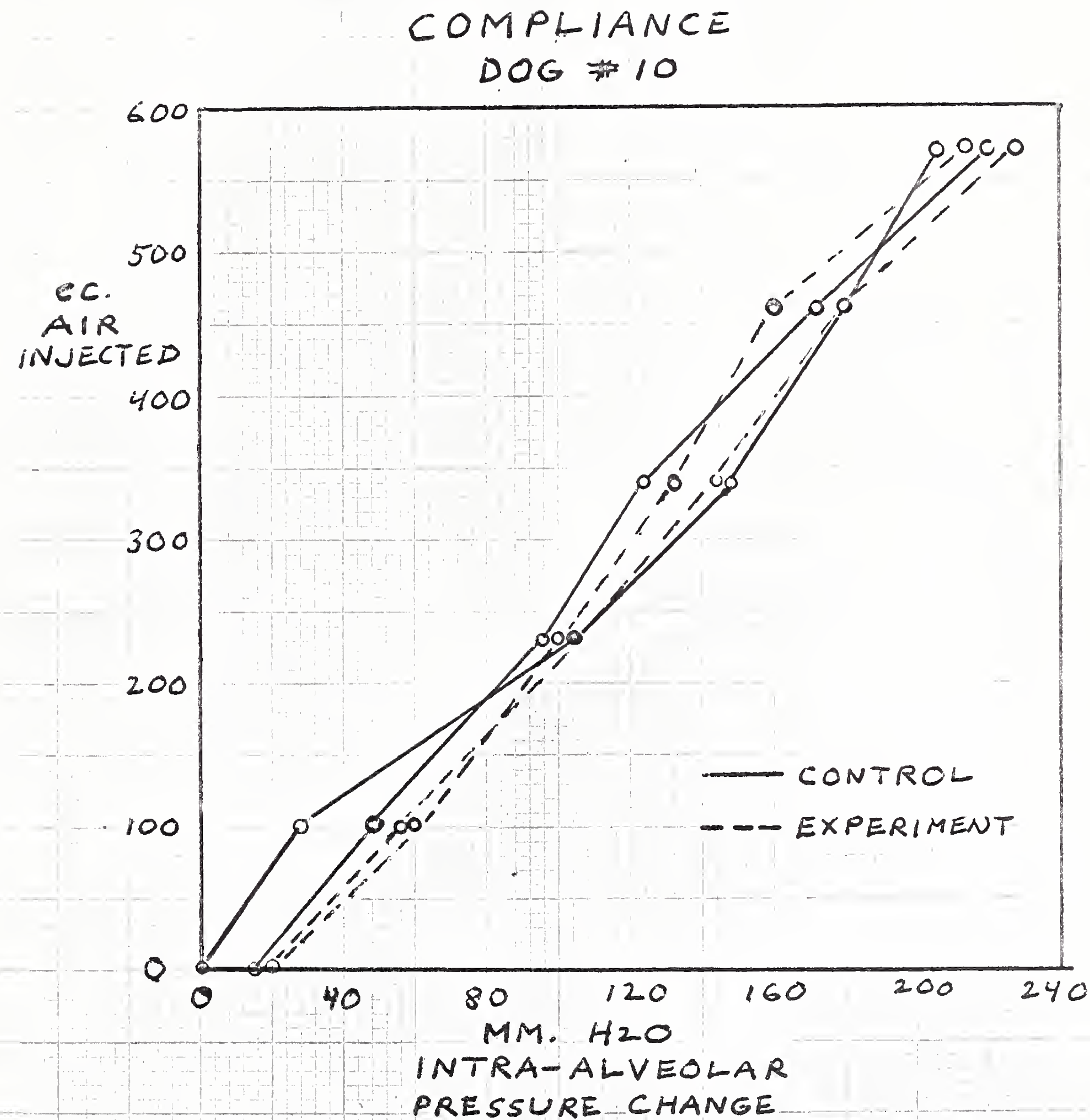

COMPLIANCE
DOG #10
600
500
400
300
200
100
0
cc.
AIR
INJECTED
0
40
80
120
160
200
240
MM. H2O
INTRA-ALVEOLAR
PRESSURE CHANGE
CONTROL
EXPERIMENT

FIGURE IX.

NO 11 PULMONARY ARTERY PRESSURE